☐ 1: Breast Cancer Res Treat 2000 Sep;63 (2):147-52

Inhibitory effects of Indole-3-carbinol on invasion and migration in human breast cancer cells.

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Indole-3-carbinol (I3C) is a promising phytochemical agent in chemoprevention of breast cancer. Our present study is the first description of I3C that significantly inhibits the cell adhesion, spreading and invasion associated with an up-regulation of PTEN (a tumor suppressor gene) and E-cadherin (a regulator of cell-cell adhesion) expression in T47-D human breast cancer cells. Therefore, I3C exhibits anti-cancer activities by suppressing breast tumor cell growth and metastatic spread. Metastatic breast cancer is a devastating problem, clinical application of I3C as a potent chemopreventive agent may be helpful in limiting breast cancer invasion and metastasis.

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1: Biochim Biophys Acta 2000 Dec 20;1498(2-3):99-111

ELSEVIER SCIENCE
FULL-TEXT ARTICLE

The biology of the receptor for advanced glycation end products and its ligands.

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Receptor for advanced glycation end products (RAGE) is a multiligand member of the immunoglobulin superfamily of cell surface molecules whose repertoire of ligands includes advanced glycation end products (AGEs), amyloid fibrils, amphoterins and S100/calgranulins. The overlapping distribution of these ligands and cells overexpressing RAGE results in sustained receptor expression which is magnified via the apparent capacity of ligands to upregulate the receptor. We hypothesize that RAGE-ligand interaction is a propagation factor in a range of chronic disorders, based on the enhanced accumulation of the ligands in diseased tissues. For example, increased levels of AGEs in diabetes and renal insufficiency, amyloid fibrils in Alzheimer's disease brain, amphoterin in tumors and S100/calgranulins at sites of inflammation have been identified. The engagement of RAGE by its ligands can be considered the 'first hit' in a two-stage model, in which the second phase of cellular perturbation is mediated by superimposed accumulation of modified lipoproteins (in atherosclerosis), invading bacterial pathogens, ischemic stress and other factors. Taken together, these 'two hits' eventuate in a cellular response with a propensity towards tissue destruction rather than resolution of the offending pathogenic stimulus. Experimental data are cited regarding this hypothesis, though further studies will be required, especially with selective low molecular weight inhibitors of RAGE and RAGE knockout mice, to obtain additional proof in support of our concept.

Publication Types:
- Review
- Review, Tutorial

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